HEART WALL TENSION REDUCTION APPARATUS

Field of the Invention

The present invention pertains to the field of apparatus for treatment of a failing heart. In particular, the apparatus of the present invention is directed toward reducing the wall stress in the failing heart.

Background of the Invention

The syndrome of heart failure is a common course for the progression of many forms of heart disease. Heart failure may be considered to be the condition in which an abnormality of cardiac function is responsible for the inability of the heart to pump blood at a rate commensurate with the requirements of the metabolizing tissues, or can do so only at an abnormally elevated filling pressure. There are many specific disease processes that can lead to heart failure with a resulting difference in pathophysiology of the failing heart, such as the dilatation of the left ventricular chamber. Etiologies that can lead to this form of failure include idiopathic cardiomyopathy, viral cardiomyopathy, and ischemic cardiomyopathy.

The process of ventricular dilatation is generally the result of chronic volume overload or specific damage to the myocardium. In a normal heart that is exposed to long term increased cardiac output requirements, for example, that of an athlete, there is an adaptive process of slight ventricular dilation and muscle myocyte hypertrophy. In this way, the

heart fully compensates for the increased cardiac output requirements. With damage to the myocardium or chronic volume overload, however, there are increased requirements put on the contracting myocardium to such a level that this compensated state is never achieved and the heart continues to dilate.

The basic problem with a large dilated left ventricle is that there is a significant increase in wall tension and/or stress both during diastolic filling and during systolic contraction. In a normal heart, the adaptation of muscle hypertrophy (thickening) and ventricular dilatation maintain a fairly constant wall tension for systolic contraction. However, in a failing heart, the ongoing dilatation is greater than the hypertrophy and the result is a rising wall tension requirement for systolic contraction. This is felt to be an ongoing insult to the muscle myocyte resulting in further muscle damage. The increase in wall stress is also true for diastolic filling. Additionally, because of the lack of cardiac output, there is generally a rise in ventricular filling pressure from several physiologic mechanisms. Moreover, in diastole there is both a diameter increase and a pressure increase over normal, both contributing to higher wall stress levels. The increase in diastolic wall stress is felt to be the primary contributor to ongoing dilatation of the chamber.

Prior art treatments for heart failure fall into three generally categories. The first being pharmacological, for

example, diuretics. The second being assist systems, for example, pumps. Finally, surgical treatments have been experimented with, which are described in more detail below.

With respect to pharmacological treatments, diuretics have been used to reduce the workload of the heart by reducing blood volume and preload. Clinically, preload is defined in several ways including left ventricular end diastolic pressure (LVEDP), or left ventricular end diastolic volume (LVEDV). Physiologically, the preferred definition is the length of stretch of the sarcomere at end diastole. Diuretics reduce extra cellular fluid which builds in congestive heart failure patients increasing preload conditions. Nitrates, arteriolar vasodilators, angiotensin converting enzyme inhibitors have been used to treat heart failure through the reduction of cardiac workload through the reduction of afterload. Afterload may be defined as the tension or stress required in the wall of the ventricle during ejection. Inotropes like digoxin are cardiac glycosides and function to increase cardiac output by increasing the force and speed of cardiac muscle contraction. These drug therapies offer some beneficial effects but do not stop the progression of the disease.

Assist devices include mechanical pumps and electrical stimulators. Mechanical pumps reduce the load on the heart by performing all or part of the pumping function normally done by the heart. Currently, mechanical pumps are used to sustain the patient while a donor heart for transplantation becomes

available for the patient. Electrical stimulation such as bi-ventricular pacing have been investigated for the treatment of patients with dilated cardiomyopathy.

There are at least three surgical procedures for treatment of heart failure: 1) heart transplant; 2) dynamic cardiomyoplasty; and 3) the Batista partial left ventriculectomy. Heart transplantation has serious limitations including restricted availability of organs and adverse effects of immunosuppressive therapies required following heart transplantation. Cardiomyoplasty includes wrapping the heart with skeletal muscle and electrically stimulating the muscle to contract synchronously with the heart in order to help the pumping function of the heart. The Batista partial left ventriculectomy includes surgically remodeling the left ventricle by removing a segment of the muscular wall. This procedure reduces the diameter of the dilated heart, which in turn reduces the loading of the heart. However, this extremely invasive procedure reduces muscle mass of the heart.

Summary of the Invention

The present invention pertains to a non-pharmacological, passive apparatus for the treatment of a failing heart. The device is configured to reduce the tension in the heart wall. It is believed to reverse, stop or slow the disease process of a failing heart as it reduces the energy consumption of the

failing heart, decrease in isovolumetric contraction, increases sarcomere shortening during contraction and an increase in isotonic shortening in turn increases stroke volume. The device reduces wall tension during diastole (preload) and systole.

In one embodiment, the apparatus includes a tension member for drawing at least two walls of the heart chamber toward each other to reduce the radius or area of the heart chamber in at least one cross sectional plane. The tension member has anchoring member disposed at opposite ends for engagement with the heart or chamber wall.

In another embodiment, the apparatus includes a compression member for drawing at least two walls of a heart chamber toward each other. In one embodiment, the compression member includes a balloon. In another embodiment of the apparatus, a frame is provided for supporting the compression member.

Yet another embodiment of the invention includes a clamp having two ends biased toward one another for drawing at least two walls of a heart chamber toward each other. The clamp includes at least two ends having atraumatic anchoring member disposed thereon for engagement with the heart or chamber wall.

Brief Description of the Drawings

Figure 1 is a transverse cross-section of the left and right ventricles of a human heart showing the placement of a splint in accordance with the present invention;

Figure 2 is a transverse cross-section of the left and right ventricles of a human heart showing the placement of a balloon device in accordance with the present invention;

Figure 3 is a transverse cross-section of the left and right ventricles of a human heart showing the placement of an external compression frame structure in accordance with the present invention;

Figure 4 is a transverse cross-section of the left and right ventricles of a human heart showing a clamp in accordance with the present invention;

Figure 5 is a transverse cross-section of the left and right ventricles of a human heart showing a three tension member version of the splint of Figure 1;

Figure 6 is a transverse cross-section of the left and right ventricles of a human heart showing a four tension member version of the splint shown in Figure 1;

Figure 7 is a vertical cross-section of the left ventricle and atrium, the left ventricle having scar tissue;

Figure 8 is a vertical cross-section of the heart of Figure 7 showing the splint of Figure 1 drawing the scar tissue toward the opposite wall of the left ventricle;

Figure 9 is a vertical cross-section of the left ventricle and atrium of a human heart showing a version of the splint of Figure 1 having an elongate anchor bar;

Figure 10 is a side view of an undeployed hinged anchor member;

Figure 11 is a side view of a deployed hinged anchor member of Figure 10;

Figure 12 is a cross-sectional view of an captured ball anchor member;

Figure 13 is a perspective view of a cross bar anchor member;

Figure 14 is a idealized cylindrical model of a left ventricle of a human heart;

Figure 15 is a splinted model of the left ventricle of Figure 14;

Figure 16 is a transverse cross-sectional view of Figure 15 showing various modeling parameters;

Figure 17 is a transverse cross-section of the splinted left ventricle of Figure 15 showing a hypothetical force distribution; and

Figure 18 is a second transverse cross-sectional view of the model left ventricle of Figure 15 showing a hypothetical force distribution.

Detailed Description of the Invention

Referring now to the drawings wherein like reference numerals refer to like elements throughout the several views, Figure 1 shows a transverse cross-section of a left ventricle 10 and a right ventricle 12 of a human heart 14. Extending through the left ventricle is a splint 16 including a tension member 18 and oppositely disposed anchors 20. Splint 16 as shown in Figure 1 has been positioned to draw opposite walls of left ventricle 10 toward each other to reduce the "radius" of the left ventricular cross-section or the cross-sectional area thereof to reduce left ventricular wall stresses. It should be understood that although the splint 16 and the alternative devices disclosed herein are described in relation to the left ventricle of a human heart, these devices could also be used to reduce the radius or cross-sectional area of the other chambers of a human heart in transverse or vertical directions, or at an angle between the transverse and vertical.

Figure 2 discloses an alternate embodiment of the present invention, wherein a balloon 200 is deployed adjacent the left ventricle. The size and degree of inflation of the balloon can be varied to reduce the radius or cross-sectional area of left ventricle 10 of heart 14.

Figure 3 shows yet another alternative embodiment of the present invention deployed with respect to left ventricle 10 of human heart 14. Here a compression frame structure 300 is

engaged with heart 14 at atraumatic anchor pads 310. A compression member 312 having an atraumatic surface 314 presses against a wall of left ventricle 10 to reduce the radius or cross-sectional area thereof.

Figure 4 is a transverse cross-sectional view of human heart 14 showing yet another embodiment of the present invention. In this case a clamp 400 having atraumatic anchor pads 410 biased toward each other is shown disposed on a wall of left ventricle 10. Here the radius or cross-sectional area of left ventricle 10 is reduced by clamping off the portion of the wall between pads 410. Pads 410 can be biased toward each other and/or can be held together by a locking device.

Each of the various embodiments of the present invention disclosed in Figures 1-4 can be made from materials which can remain implanted in the human body indefinitely. Such biocompatible materials are well-known to those skilled in the art of clinical medical devices.

Figure 5 shows an alternate embodiment of the splint of Figure 1 referred to in Figure 5 by the numeral 116. The embodiment 116 shown in Figure 5 includes three tension members 118 as opposed to a single tension member 18 as shown in Figure 1. Figure 6 shows yet another embodiment of the splint 216 having four tension members 218. It is anticipated that in some patients, the disease process of the failing heart may be so advanced that three, four or more tension members may be desirable to reduce the heart wall stresses

more substantially than possible with a single tension member as shown in Figure 1.

Figure 7 is a partial vertical cross-section of human heart 14 showing left ventricle 10 and left atrium 22. As shown in Figure 7, heart 14 includes a region of scar tissue 24 associated with an aneurysm or ischemia. As shown in Figure 7, the scar tissue 24 increases the radius or cross-sectional area of left ventricle 10 in the region affected by the scar tissue. Such an increase in the radius or cross-sectional area of the left ventricle will result in greater wall stresses on the walls of the left ventricle.

Figure 8 is a vertical cross-sectional view of the heart 14 as shown in Figure 7, wherein a splint 16 has been placed to draw the scar tissue 24 toward an opposite wall of left ventricle 10. As a consequence of placing splint 16, the radius or cross-sectional area of the left ventricle affected by the scar tissue 24 is reduced. The reduction of this radius or cross-sectional area results in reduction in the wall stress in the left ventricular wall and thus improves heart pumping efficiency.

Figure 9 is a vertical cross-sectional view of left ventricle 10 and left atrium 22 of heart 14 in which a splint 16 has been placed. As shown in Figure 9, splint 16 includes an alternative anchor 26. The anchor 26 is preferably an elongate member having a length as shown in Figure 9 substantially greater than its width (not shown). Anchor bar

26 might be used to reduce the radius or cross-sectional area of the left ventricle in an instance where there is generalized enlargement of left ventricle 10 such as in idiopathic dilated cardiomyopathy. In such an instance, bar anchor 26 can distribute forces more widely than anchor 20.

Figures 10 and 11 are side views of a hinged anchor 28 which could be substituted for anchors 20 in undeployed and deployed positions respectively. Anchor 28 as shown in Figure 10 includes two legs similar to bar anchor 26. Hinged anchor 28 could include additional legs and the length of those legs could be varied to distribute the force over the surface of the heart wall. In addition there could be webbing between each of the legs to give anchor 28 an umbrella-like appearance. Preferably the webbing would be disposed on the surface of the legs which would be in contact with the heart wall.

Figure 12 is a cross-sectional view of a capture ball anchor 30. Capture ball anchor 30 can be used in place of anchor 20. Capture ball anchor 30 includes a disk portion 32 to distribute the force of the anchor on the heart wall, and a recess 34 for receiving a ball 36 affixed to an end of tension member 18. Disk 32 and recess 34 include a side groove which allows tension member 38 to be passed from an outside edge of disk 32 into recess 34. Ball 36 can then be advanced into recess 34 by drawing tension member 18 through an opening 38 in recess 34 opposite disk 32.

Figure 13 is a perspective view of a cross bar anchor 40. The cross bar anchor 40 can be used in place of anchors 20. The anchor 40 preferably includes a disk or pad portion 42 having a cross bar 44 extending over an opening 46 in pad 42. Tension member 18 can be extended through opening 46 and tied to cross bar 42 as shown.

In use, the various embodiments of the present invention are placed in or adjacent the human heart to reduce the radius or cross-section area of at least one chamber of the heart. This is done to reduce wall stress or tension in the heart or chamber wall to slow, stop or reverse failure of the heart. In the case of the splint 16 shown in Figure 1, a canula can be used to pierce both walls of the heart and one end of the splint can be advanced through the canula from one side of the heart to the opposite side where an anchor can be affixed or deployed. Likewise, an anchor is affixed or deployed at the opposite end of splint 16.

Figure 14 is a view of a cylinder or idealized heart chamber 48 which is used to illustrate the reduction of wall stress in a heart chamber as a result of deployment of the splint in accordance with the present invention. The model used herein and the calculations related to this model are intended merely to illustrate the mechanism by which wall stress is reduced in the heart chamber. No effort is made herein to quantify the actual reduction which would be realized in any particular in vivo application.

Figure 15 is a view of the idealized heart chamber 48 of Figure 14 wherein the chamber has been splinted along its length L such that a "figure eight" cross-section has been formed along the length thereof. It should be noted that the perimeter of the circular transverse cross-section of the chamber in Figure 14 is equal to the perimeter of the figure eight transverse cross-section of Figure 15. For purposes of this model, opposite lobes of the figure in cross-section are assumed to be mirror images.

Figure 16 shows various parameters of the Figure 8 cross-section of the splinted idealized heart chamber of Figure 15. Where ℓ is the length of the splint between opposite walls of the chamber, R_2 is the radius of each lobe, θ is the angle between the two radii of one lobe which extends to opposite ends of the portion of the splint within chamber 48 and h is the height of the triangle formed by the two radii and the portion of the splint within the chamber 48 (R_1 is the radius of the cylinder of Figure 14). These various parameters are related as follows:

$$h = R_2 \cos(\theta/2)$$

$$\ell = 2 R_2 \sin(\theta/2)$$

$$R_2 = R_1\pi / (2\pi - \theta)$$

From these relationships, the area of the figure eight cross-section can be calculated by:

$$A_2 = 2\pi(R_2)^2 (1-\theta/2\pi) + h\ell$$

Where chamber 48 is unsplinted as shown in Figure 14 A_1, the original cross-sectional area of the cylinder is equal to A_2 where $\theta = 180°$, $h = 0$ and $\ell = 2R_2$. Volume equals A_2 times length L and circumferential wall tension equals pressure within the chamber times R_2 times the length L of the chamber.

Thus, for example, with an original cylindrical radius of four centimeters and a pressure within the chamber of 140 mm of mercury, the wall tension T in the walls of the cylinder is 104.4 newtons. When a 3.84 cm splint is placed as shown in Figures 15 and 16 such that $\ell = 3.84$ cm, the wall tension T is 77.33 newtons.

Figures 17 and 18 show a hypothetical distribution of wall tension T and pressure P for the figure eight cross-section. As θ goes from 180° to 0°, tension T_s in the splint goes from 0 to a 2T load where the chamber walls carry a T load.

It will be understood that this disclosure, in many respects, is only illustrative. Changes may be made in details, particularly in matters of shape, size, material, and arrangement of parts without exceeding the scope of the invention. Accordingly, the scope of the invention is as defined in the language of the appended claims.